Low Infection Types Produced by *Puccinia graminis* f. sp. *tritici* and Wheat Lines with Designated Genes for Resistance

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ABSTRACT

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Between 72 and 120 wheat lines were tested annually from 1970 through 1977, to characterize the reactions of various specific resistances to stem rust caused by 100 to 300 cultures of *Puccinia graminis* f. sp. *tritici* that represented the ranges of pathogen virulence detected in the annual race survey. Annually, 10 to 100 cultures also were retested. The wheat genotypes that were studied can be classified in three groups based on responses to North American rust fungus cultures: (i) those susceptible to all or nearly all of the cultures studied, ie, *Sr*9f, 9g, 16, 18, 19, 20, 28, LC, McN, and Kt'2'; (ii) those differential in response, ie, *Sr*5, 6, 7a, 7b, 8, 9a, 9b, 9d, 9e, 10, 11, 12, 14, 15, 17, 21, 23, Tt-1, Tt-3, Tmp, dp-2, and X; and (iii) those "universally" resistant or nearly so, ie, *Sr*13, 22, 24, 25, 26, 27, 29, 30, Tt-2, and Gt. Although the low infection types produced by a range of avirulent cultures on a host with the corresponding gene for resistance over a range of environmental conditions generally were similar, numerous exceptions were observed.

Additional key words: race-specific resistance, stem rust, *Triticum*, vertical resistance.

Since 1970, the Cereal Rust Laboratory has expanded the use of recently developed host material and techniques in cereal rust research. The standard host differentials selected by Stakman et al (39) for identifying races of the wheat stem rust fungus (*Puccinia graminis* Pers. f. sp. *tritici*) were selected from a group of wheat cultivars that responded differentially to a group of cultures available from 1915 through 1922. Over the years many important new genes for stem rust resistance in wheat (*Triticum* sp.), that have been used in the development of commercial cultivars were not present in the standard differentials. For physiologic race identification to be useful, host selections possessing these resistances were added as supplemental differentials. The supplemental differentials differed between laboratories, and different sets of supplemental differentials have been used for different standard races in the same laboratory. Because some supplemental differential cultivars, such as Selkirk (CI 13100), have several genes for seedling resistance to wheat stem rust, ie, *Sr* genes 6, 7b, 9d, 17, and 23 (32), the race designations were useful only to categorize pathogen virulence against that cultivar; ie, generally, the reaction of another host cultivar to a given culture could not be predicted. Therefore, the material used for identifying pathogen races was changed to a series of host lines, each with a single designated gene for wheat stem rust resistance. It was expected that this change would provide continuity without frequent changes of supplemental differential hosts, and would allow estimation of the annual changes in pathogen virulence frequency for wheat cultivars in the USA.

The low infection type (LIT) that results from the interaction between the wheat stem rust pathogen and designated genes for resistance in the host have been reported independently (see Table 3 for references). Most reports contain results of testing one or a few isolates of the pathogen and a few host lines with single genes for resistance in a common background. Host background and environmental conditions affect the expression of the interaction (26). Utilization of the infection type to postulate virulence genes in the pathogen and resistance genes in the host requires recognition of the range of the low infection types produced by cultures and hosts with different genetic backgrounds.

This study had three objectives: to determine the range of the low infection types produced by a large number of cultures on a single host line with a specified gene for resistance, to study the variation in infection types with the same resistance gene in a few different host backgrounds, and to determine the potential use for virulence

frequency studies of various host lines with specified genes for resistance.

MATERIALS AND METHODS

Since 1970, over 1,600 cultures from annual *P. graminis* f. sp. *tritici* race surveys in Mexico, the USA, and in adjacent Canada have been tested on a variable number of host lines, including the standard and supplemental differential cultivars, wheat lines with a "single" gene for resistance in various backgrounds, the background lines without the donated gene, and lines with known combinations of genes for resistance. Cultures were selected for differences in virulence combinations on the standard and supplemental differentials in 1970 and 1971. In 1972, cultures were selected for virulence combinations on *Sr*5, 6, 7b, 8, 9a, 9d, 9e, and 11. In 1973, *Sr*9b, 10, 13, and Tt-1 were added to the list, and in 1974, *Sr*15, 16, 17, and Tmp were added. Annually, cultures from each state were studied from which a given virulence combination (race) was obtained in the annual race survey. Also, each year 10 to 100 cultures tested in previous years were retested.

Cultures were removed from storage and inoculated to a susceptible host that had been treated with maleic hydrazide to enhance spore production. Sufficient uredospores were collected after 14 to 19 days to inoculate six to 10 seedlings of 72 to 120 different host lines. Plants were grown in vermiculite and fertilized 5 and 8 days after planting with a water-soluble fertilizer (23-19-17, N-P-K) at a rate of 2.5 gm per 24 host lines. The 7-day-old plants were inoculated with a spore suspension in a light weight mineral oil carrier, then placed in a dew chamber at 18 C overnight. The next morning the chamber was illuminated (10,000 lux) and the temperature rose gradually to 30 C over a 4 hr period so the dew evaporated slowly. Then the plants were placed in a greenhouse environment at 18 C supplemented with 11,000 lux of fluorescent light. In a few trials, plants were grown in growth chambers at selected temperatures. Infection types (39) were recorded 13 days after inoculation. Briefly, infection types are based on lesion characteristics, 0 = immune, ; = hypersensitive flecks, 1 = minute uredia, 2 = small uredia, 3 = medium uredia, X = variable sized uredia, 4 = large uredia (host susceptible). Plus and minus indicate variation within infection type; C and N indicate pronounced chlorosis or necrosis, respectively. Three variations of standard infection-type recording procedures (39) were used: a comma was used to separate discrete infection types on a single leaf, two infection types written together (ie, 23 or 0;) indicated continual variation between the two on a single leaf, and the infection types were written in decreasing order of frequency.

TABLE 1. Number of wheat lines tested and number of avirulent cultures of *Puccinia graminis* f. sp. *tritici* used to evaluate the variation in low infection type due to variation in host and pathogen genotypes

Sr gene	Number of Lines with *Sr* gene	Number of Cultures avirulent	*Sr* gene	Number of Lines with *Sr* gene	Number of Cultures avirulent
5	35	231	20	4	94
6	29	1,161	21	2	223
7a	16	391	22	3	1,256
7b	35	654	23	4	82
8	16	592	24	3	1,499
9a	11	832	25	3	885
9b	15	1,185	26	2	688
9d	10	250	27	1	688
9e	13	1,281	28	2	82
9f	12	49	29	3	1,054
9g	5	1	30	3	1,097
10	11	600	Tt-1	12	867
11	30	1,044	Tt-2	3	1,650
12	8	284	Tt-3	3	117
13	10	1,673	Tmp	2	1,279
14	5	626	LC	10	52
15	6	356	Gt	3	193
16	10	195	dp-2	10	1,067
17	11	754	X	23	80
18	6	56	McN	1	34
19	7	43	Kt'2'	3	155

RESULTS AND DISCUSSION

The data generated by these experiments are too voluminous to be published in entirety. Table 1 lists the host genes tested, the number of host lines tested that had that gene either alone or in combination with other genes, and the number of test cultures avirulent on lines with each resistance gene. Many of the host lines had more than one resistance gene; therefore, not all infection types expressed were that of the designated host-pathogen gene pair. Thus, due to epistasis, when a host with both *Sr*6 and 8 was inoculated with a culture avirulent on both gene pairs, the resultant LIT was that of *Sr*6. Table 2 lists host lines that, when inoculated with North American cultures, resulted in an infection type that almost always was due to the designated host-gene pathogen pair. These are the recommended sources of host material to determine pathogen virulence. The mean LIT is recorded for tests done in the winter at 18 C, but when this LIT differed from the LIT produced at higher temperatures the latter also are given in the text section for that gene.

Sr5. Each culture that was avirulent on lines with *Sr*5 produced a LIT which was characteristic for that host line. However, some differences in LIT were observed among different *Sr*5-bearing host lines infected with a single culture. The extremes in our tests were represented by ISr5-Ra and LCSr5R1, with LITs of 0; and 0, respectively. These infection types were unaffected by temperature in the range of conditions and host and pathogen genotypes that were studied.

Gene *Sr*5 for resistance to stem rust was present in cultivars Reliance and Frontana-Kenya 58-Newthatch (FKN) of the standard or supplemental differentials, respectively, and the 0 or 0; infection types recorded for standard races 9, 17, 19, 21, 24, 29, and 49 on cultivar Reliance were due to this gene. In the USA, *Sr*5 was effective against 70% of the cultures in 1921 and 1922 and 65% in 1941. Between 1918 and 1966, over 50% of the cultures have been virulent on *Sr*5, and since 1966 over 90% have been virulent. Avirulence on lines with *Sr*5 was associated with standard races 17, 19, 21, and 29 in 1921; races 9, 17, and 21 in 1922; and race 17 in 1942. Gene *Sr*5 is present in many U.S. wheat cultivars.

Sr6. The LIT symptoms varied with temperature and ranged from a fleck at a constant 18 C, through a chlorotic mesothetic reaction, to a fully susceptible infection type at 30 C. However, with day and night variations in temperature, a gradual change from hypersensitivity to susceptibility was more closely associated with the minimum temperature than the maximum temperature. Owing to temperature sensitivity we are unsure if lines or cultures regularly vary in infection type. However, we have a few cultures of recent aecial origin that regularly produced a 1N LIT at 18 C on ISr6-Ra but were virulent on Line D sel. This LIT may be the result of the presence of gene *Sr*U which was by Loegering (21).

Gene *Sr*6 is not present in the standard differentials, but is in the supplemental differential cultivars Selkirk, Bowie, and FKN. *Sr*6 is a valuable differential in North America, and occurs in some of the wheat cultivars grown in the northern part of the USA. The temperature sensitivity of *Sr*6 limits its value as a differential, but useful results were obtained when night temperatures ranged 18–23 C and maximum temperatures were below 30 C. From 6 to 26% of the USA cultures identified annually from 1972 through 1977 were virulent on *Sr*6.

Sr7a. The LIT ranged from 13-C to a 23C. Part of this variation was due to host background. The expressed LIT of *Sr*7a in a Marquis background was 13-C, but when *Sr*7a was transferred to a background with fewer genes for vertical resistance, the LIT was a 2C to a 3C. With some cultures the chlorosis was less evident and with others the size of the sporulation area varied. These variations masked any temperature effects on the interaction.

Under our current experimental conditions LITs were too difficult to assess to permit the use of any *Sr*7a-bearing plant line as a differential cultivar. *Sr*7a occurred in the supplemental

TABLE 2. Wheat lines and cultivars selected as representative for the *Sr* genes they possess and currently recommended for determining virulence of *Puccinia graminis* f. sp. *tritici*

Sr gene	Low infection type[a]	Line or cultivar	CI[b]	Originator or source of seed
5	0;	ISr5-Ra	14159	W. Q. Loegering
	0	LCSr5Rl(Rl-B sel)		N. D. Williams
6	;	ISr6-Ra	14163	W. Q. Loegering
	;	Line D sel		Univ. of Sydney[c]
7a	23C	Line G sel		Univ. of Sydney
	13-C	MqSr7aEg101 sel	15083	D. R. Knott
7b	2	ISr7b-Ra	14165	W. Q. Loegering
	2	LCSr7bMq sel		N. D. Williams
8	2	ISr8-Ra	14167	W. Q. Loegering
	2	Mentana	12448	Cultivar
9a	2−,23	ISr9a-Ra	14169	W. Q. Loegering
	2−,23	sr6sr8Sr9a	14183	W. Q. Loegering
9b	22+	W2691Sr9b (Line AA sel)	17386	A. P. Roelfs
	22+	MqSr9bKy117A sel	17783	D. R. Knott
9d	;2−	ISr9d-Ra	14177	W. Q. Loegering
	;1	Mindum	5296	USA cultivar
9e	;1+	Vernstein		Univ. of Sydney
	;1	Vernal	3686	Cultivar
9f	;2−	Sr6sr8sr9a	14188	W. Q. Loegering
9g	2−	Acme	5284	USA cultivar
	2−	Kubanka	2094	USSR cultivar
10	;1+N	W2691Sr10 (Line F sel)	17388	A. P. Roelfs
	2CN	LCSr10 (Ag-4 sel)	17476	F. J. Gough
11	;2−,2+3−	ISr11-Ra	14171	W. Q. Loegering
	12−,2+3−	Line Ag (sel)		Univ. of Sydney
12	X−	Line R (sel)		Univ. of Sydney
	0;1+	BtSr12Tc (Baart/Line R)	17783	A. P. Roelfs
13	2+3	W2691Sr13 (Line S sel)	17387	A. P. Roelfs
	2+3	MqSr13Khp	15088	D. R. Knott
14	23C	Line A sel		Univ. of Sydney
	12C	MqSr14Khp	15089	D. R. Knott
15	;1+N	Line AB sel		Univ. of Sydney
	;1+N	Norka		Austral. cultivar
16	2	ISr16-Ra	14173	W. Q. Loegering
	2	ITha3B-Ra	14175	W. Q. Loegering
17	;1N	Combination VII		Univ. of Sydney
	X−N	LCSr17KH		F. J. Gough
18	;2=	LCSr18Mq (Mq-A sel)		N. D. Williams
	;2=	LCSr18R1 (R1-A sel)		N. D. Williams
19	1−	LCSr19Mq (Mq-B sel)		N. D. Williams
20	2=	LCSr20Rl (Rl-C sel)		N. D. Williams
21	1−2−	*Triticum monococcum* L. derivative		Univ. of Sydney
	;1=	Einkorn	2433	Cultivar
22	0;	*Triticum boeoticum* Boiss.		Univ. of Sydney
	2;	*Triticum boeoticum* derivative		Univ. of Sydney
23	23C	Exchange	12635	English cultivar

(continued)

TABLE 2. (continued)

Sr gene	Low infection type[a]	Line or cultivar	CI[b]	Originator or source of seed
24	2+2– 2+2–	LCSr24Ag (Ag-2 sel) BtSr24Ag (Baart/Ag-2)	17474	F. J. Gough A. P. Roelfs
25	2 2–	LCSr25Ars (Ars-3 sel) Agrus	17473 13228	F. J. Gough USA cultivar
26	;2– ;2–	Eagle Line U	PI 365582	Austral. cultivar D. R. Knott
27	0;	Wheat-Rye Translocation, WRT 238-5	14141	A. C. Acosta
28	; ;	Line AD sel Kota	5878	Univ. of Sydney USA cultivar
29	2+2– 2+2–	Etiole de Choisy Pusa 4/Etiole de Choisy	PI 193108	French cultivar Algeria
30	22++ 2–2	BtSr30Wst Festiguay	PI 330957	B. Skovmand Austral. cultivar
Tt-1	0, 0;1+, 40; 0, 0;1+, 40;	W2691SrTt-1 (Line C sel) Idaed 59	17385 13631	A. P. Roelfs USA cultivar
Tt-2	0;	Line W sel		Univ. of Sydney
Tt-3	0,;1+C 0,;1+C	Fed/SrTt-3 Fed*2/SrTt-3		Univ. of Sydney Univ. of Sydney
Tmp	2– 2–	Triumph 64 Trison	13679 17278	USA cultivar USA cultivar
LC	2– 2–	Little Club Baart	4066 1697	Cultivar Austral. cultivar
Gt	2+ 2	BtSrGtGt Gamut	PI 329230	A. P. Roelfs Austral. cultivar
dp-2	2 2++	Medea Ap9d Golden Ball deriv. sel	3255	Tunisia Univ. of Sydney
X	23C 23C	Marquis Prelude *8/Marquis	3641	Canadian cultivar P. L. Dyck
McN	2–;	McNair 701	15288	USA cultivar
Kt'2'	2 2	Line AE sel Kota	5878	Univ. of Sydney USA cultivar

[a] Low infection type observed at 18 C. Comma separates infection variation due to culture tested.
[b] CI = cereal investigation; PI = plant introduction.
[c] University of Sydney team (J. Gyarfas, N. H. Luig, R. A. McIntosh, T. T. The, and I. A. Watson).

differential cultivars Kenya Farmer and FKN, and possibly in the standard differential cultivar Khapli. The variation in LITs produced in plants with gene *Sr*7a also made it difficult to define percent virulence in the pathogen or to determine the distribution of this resistance gene in commercial cultivars.

***Sr*7b.** The LIT associated with *Sr*7b was a 2 and it was easy to recognize unless infection density was high. The infection type was relatively stable among cultures, host backgrounds, and temperatures, although a few cultures of recent aecial origin produced LIT 2++.

The *Sr*7b gene was present in Marquis and Kota of the standard differentials, and Selkirk of the supplemental differentials. More than 73% of the USA isolates have been virulent on *Sr*7b since 1918 except for 1972 and 1977 when 72% and 69%, respectively, were virulent. The principal races avirulent on *Sr*7b were 151-QCB, -QFB, and -QSH. Distribution of this gene in the USA wheat population is limited.

***Sr*8.** The characteristic LIT was a 2, with no variation due to culture, temperature, or host background. This gene was present in cultivars Bowie and FKN of the supplemental differentials. Currently 75–90% of the isolates are virulent on lines with *Sr*8. Before 1970 avirulence was much more common; most isolates of race 15 and race 56 were avirulent on *Sr*8.

***Sr*9a.** Two distinct LITs occurred depending on the cultures used. One LIT typified by infection with race 15-TLM (36) was a 2–; the other, which was typified by infection with race 151-QSH, was a 23. These LITs seemed to be temperature insensitive. These results are based on extensive testing on *Sr*9a in both the Marquis and Chinese backgrounds.

The host gene, *Sr*9a, did not occur in the standard or supplemental differentials. Since 1972, 10–45% of the population in the USA has been virulent on *Sr*9a. The current races 15-TNM,

-TDM, -TLM, and 151-QSH were avirulent.

***Sr*9b.** The LIT was a 2 to a 2+ at 18 C, while at 30 C the LIT varied from a 2− to a 2. The LIT was recognizable only by the size of the sporulation area; the lesion was approximately the same size as that of the high infection type, and no chlorosis was associated with the lesion. An exception was found; race 56-MBC and a few cultures obtained from aecial spreads frequently caused a lesion with some associated purple color. No major differences were noted in LIT with different host backgrounds.

The gene *Sr*9b did not occur in the standard differentials but was present in cultivars Kenya Farmer, and FKN. This gene has been effective against most cultures since 1972. The proportion of the population with virulence varied from 4% in 1974 to 13% in 1973. Races 151-QSH; 113-RKQ, -RTQ, and -RPQ; and 11-RHR and -RCR comprise the majority of the cultures that were virulent on *Sr*9b. Probably this gene was brought into the commercial cultivars from the Kenya wheats; however, the difficulty in recognizing the LIT makes detection of *Sr*9b in an unknown background difficult.

***Sr*9d.** The LIT was a fleck to a 2−, but it varied unexplainably. We did not find this to be associated with either host line or pathogen culture. The LIT was relatively unaffected by temperature.

The gene *Sr*9d was present in the standard differential cultivars Arnautka, Mindum, and Spelmar. The proportion of the pathogen population with virulence on lines with *Sr*9d was lowest in 1926 (35%), 1934 (36%), 1937 and 1938 (31%), 1947 (28%), 1960 (32%), and 1962 (39%). The lack of virulence in the population was due to a predominance of races 18 and 34 in 1926; 34, 36, and 56 in 1934; and 56 in 1937, 1938, 1947, 1960, and 1962. Since 1972, over 96% of the isolates made in the USA have been virulent on lines with *Sr*9d.

***Sr*9e.** The LIT was a fleck to a 1+. The infection type remained relatively stable over a wide range of temperatures, pathogen cultures, and host backgrounds. However, the LIT tended to be higher if *Sr*9e was in a hexaploid rather than a tetraploid background. In testing Vernstein with certain cultures of aecial origin a Y (a modified X infection type with the largest uredia toward the tip of the leaf and the smaller toward the base of the leaf) LIT was obtained. These cultures were virulent on the other sources of *Sr*9e used in these tests. We have temporarily designed this host gene *Sr*Vrn. It was inherited as a dominant gene; however, the LIT was a 2 in the F_1.

The gene *Sr*9e occurred in Vernal and Yuma of the standard and supplemental differentials, respectively. Virulence on lines with *Sr*9e was low until 1950, when race 15 increased and became a major race. Five to 14% of the isolates were virulent on *Sr*9e from 1918 through 1922, and only 1% were virulent in 1925, 1928, and 1931. Since 1972, from 54 to 86% of the isolates obtained in the USA have been virulent on lines with *Sr*9e. Many of the USA

TABLE 3. Genes for resistance to wheat stem rust, the host in which they originally were described, the reported low-infection types, and the average low-infection type(s) observed in tests with over 1,000 cultures of *Puccinia graminis* f. sp. *tritici*, and several host backgrounds

Sr gene	Reference	Cultivar in which *Sr* gene originally was reported	Low infection types[b]	
			Reported	Observed mean
5	1,34	Kanred	0	0,0;
6	18	McMurchy, Kenya 58, Red Egyptian	;	;*
7a	18	Egypt NA 95, Kenya 58, Kenya 117A	1+	13−C,23C
7b	21	Hope	2−	2
8	18	Red Egyptian	2+−	2
9a	18	Egypt NA 95, Red Egyptian	2+−	2−,23
9b	18	Kenya 117A	2+−	22+
9d(1)	6,24	Hope		;2−
9e	28	Vernstein	;	;1+
9f	22	Chinese		;2−
9g	23	Thatcher, Kubanka, Acme	2−2,2+3−	2−
10	18	Kenya 117A, Egypt NA 95	1+−2	;1−N,2CN*
11	18	Gabo, Lee, Timstein	1−1+	;2−,2+3−
12	38	Thatcher		0;X−
13	16	Khapstein	2−3	2+3*
14	16	Khapstein	2+−4N	13C
15	41	Norka		;1+N*
16	22	Thatcher		2
17	33	Hope	X−N	;1N,X−N*
18	3,7	Many wheats	0;2=	;2=
19	3	Marquis	1+−	1−
20	3	Marquis, Reliance	2−3	2=
21	38	Einkorn		;1=
22	11,40	*Triticum boeoticum*		0;2
23	32	Selkirk, Exchange, Warden, Etiole de Choisy	1+3CN	23C
24	9,30	*Agropyron elongatum*	0;1,3=	2+2−
25	9,30	*Agropyron elongatum*	3−	22−
26	14	*Agropyron elongatum*	2−	;2−
27	2	Imperial rye		0;
28	25	Kota	0;	;
29	8,29,43	Etiole de Choisy	2−3	2+2−
30	19	Webster		2−2++
Tt-1	31	*Triticum timopheevii*	0;	0,0;1+,4;
Tt-2	31	*Triticum timopheevii*	0;1N	0;
Tt-3	31	*Triticum timopheevii*		0,;1+C
Tmp	36	Triumph 64		2−
LC	25	Little Club		2−
Gt	35	Gamut		22+
dp-2	10,37	Golden Ball		2,2++
X	...	Marquis		23C
McN	...	McNair 701		2−;
Kt'2'	...	Kota	2	2

[a] Temperature-sensitive response.

[b] Comma used to separate low-infection differences due to host background or among pathogen cultures.

durum cultivars have *Sr*9e.

***Sr*9f**. This gene resulted in a LIT of fleck to 2−. It was ineffective against all cultures tested except a few of recent aecial origin. This gene was of interest because it occurs in cultivar Chinese Spring and therefore in most of the ISr lines used as a background for many of the other host genes studied.

***Sr*9g**. This gene is in Kubanka and Acme durums and in Thatcher and Lee bread wheats (25). The LIT was reported to range from a 2− to a 2+3− depending on the culture used (25). The only avirulent culture we have is among the progeny of a cross between race 111 and 36. The LIT was a 2 with this culture.

***Sr*10**. The LIT was a ;1+N at 18 C on W2691Sr10. Under summer greenhouse temperatures the LIT type was a 1−3−CN. The LIT at 18 C was a 2CN on host line LCSr10. No change was noted in LITs when different cultures were used.

Gene *Sr*10 occurred only in Kenya Farmer of the supplemental differentials. Since 1972, 77 to 91% of the isolates have been virulent on cultivars with *Sr*10. Virulence was present in race 15-TNM, -TLM, -TDM; 151-QSH, 11-RHR, -RCR, 56-MBC, etc. The gene probably is not widespread in the wheats of the Great Plains, but it does occur frequently in the spring wheats developed in the western states and in the cultivars from Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT).

***Sr*11**. Usually the LIT for this gene was a ;2−. However, differences were noted between Line AG and ISr11-Ra, with the latter usually expressed as a ;2− and the former as a 12−. Races 15-TDM and -TBM were exceptions in that the LIT for these races was a 2 to 2+3− regardless of the host line being tested. All LITs were relatively unaffected by temperature.

Only Lee and Kenya Farmer of the supplemental differentials had *Sr*11. Since 1972, 63–83% of the isolates have been virulent on *Sr*11. Virulence was found in races 15-TNM, -TLM, 151-QSH, and 113-RTQ. Currently the gene is found in some northern hard red spring wheats as well as in many CIMMYT cultivars.

***Sr*12**. This gene was the *Sr*12 described by Sheen and Snyder (38) and not the gene described by Knott and Anderson (18). The LIT normally varied from a 0; to X−, although we have a few cultures that consistently incite an immune response. Lines homozygous for *Sr*12 that varied in LIT from 0 to 2 were observed among the progeny of a Baart/Line R cross. Probably this variation is due to differences in genetic background of the host. No tests were performed with this host material at temperatures greater than 18 C.

Gene *Sr*12 provided resistance to race 56-MBC. This gene occurs in Thatcher and its derivatives and thus is widespread in the spring wheat area. Since 1966, virulence on *Sr*12, has occurred in 91–99% of isolates in the USA.

***Sr*13**. The LIT was a 2− at 30 C, 2+3 at 18 C, and was extremely difficult to recognize unless compared with the susceptible background line. The lesion tended to be slightly smaller and the sporulating area smaller and more rounded than on the susceptible background line without *Sr*13. No difference in LIT was found between cultures. However, in the single plant line with tetraploid background that was tested at 18 C the infection type was a 2 to a 2−, much more resistant than any of the hexaploid hosts.

***Sr*14.** The LIT was a 12C to a 23C. Some variation in LIT was noted with different host backgrounds. This host gene was not tested at temperatures above 18 C. No difference was noted in LIT between cultures, except when cultures avirulent on *Sr*7b were tested, no LITs above 2C were observed for our line with *Sr*14. This result was so constant that crosses were made to determine if *Sr*7b was present in these *Sr*14 lines.

The gene *Sr*14 provides resistance to most races other than 15-TNM, -TDM, -TLM, and 56-MBC. This should be a useful differential only if a single gene is present in our lines. We have not found *Sr*14 in commercial wheats.

***Sr*15**. The LIT was a ;1+N at 18 C, but was 3N at 21 C and 44C at 23 C. A few lesions had narrow chlorotic rings around the sporulation area at 23 C. With this extreme variation in LIT due to temperature it was impossible to evaluate changes in LIT due to differing cultures and host backgrounds.

The gene *Sr*15 was not found in previously used differentials. It provided resistance against 61 to 76% of the isolates of stem rust obtained in the USA from 1975 through 1977. However, it is effective only at temperatures below 21 C, and we know of no commercial cultivars with this resistance. It may be of value in cool areas where stem rust can overwinter. Avirulence is found in races 15-TNM, -TLM, and -TDM.

***Sr*16**. The LIT for this gene was a 2. No variation due to culture, host, or environment was found.

***Sr*17**. The LIT for this gene varied from ;1N to X-N. Lines tested in a Little Club background generally were more susceptible at all temperatures than was the Combination VII line. This was due in part to Combination VII having *Sr*13 in addition to *Sr*17. Frequently the LIT produced on Combination VII was a 0; while the reaction with the same culture on LCSr17KH was X−. This infection type was somewhat temperature sensitive, with a 0;N being expressed at 18 C and a ;1+N at 23 C. A few cultures of recent aecial origin produced a 1+ infection type at 18 C.

Gene *Sr*17 occurred in the supplemental cultivar Selkirk. Part of the seedling resistance of Selkirk historically attributed to *Sr*6 probably was due to *Sr*17. Prior to the mid-1950s races 56 and 15 were virulent on lines with *Sr*17. Currently 18–44% of the USA isolates are avirulent on lines with *Sr*17. This avirulence occurs in most of the isolates of races 15-TNM, -TDM, 113-RKQ, and -RTQ. The *Sr*17 allele is widespread in USA and CIMMYT cultivars.

***Sr*18**. The LIT was a ;2=. Avirulence for this gene is rare except in aecial cultures that originate from crosses between pathogen races. Thus, little material was available for studying differences among cultures. Further, a few avirulent cultures or host lines without other resistances were available. Our interest in *Sr*18 was mainly due to its widespread occurrence in many wheat cultivars (7).

***Sr*19.** The LIT was a 1−. Avirulence was rare among the cultures tested and few host lines were available to study the effect on the Lit produced. No attempt was made to study the effects of temperature on expression of this gene.

***Sr*20**. The LIT was a 2=. Avirulence for *Sr*20 was rare, and no attempt was made to determine the variation in LIT.

***Sr*21**. The LIT was expressed as ;1= in Einkorn, a diploid wheat, but generally varied from a 1− to a 2− in the hexaploid line tested. No variation of LIT was noted between cultures or for the temperature range studied.

Einkorn was the only standard differential with *Sr*21. *Sr*21 was most effective in the USA in 1934, 1935, 1937, 1938, 1960, and 1962 when 26, 37, 36, 38, 33, and 39%, respectively, of the isolates were virulent on lines with *Sr*21. Avirulence was due primarily to races 34 and 56 in 1934 and 1935, and to race 56 since then. Since 1969, 99–100% of the cultures have been virulent on lines with *Sr*21. We have not found *Sr*21 in any commercial cultivars grown in the USA.

***Sr*22**. The LIT for this gene in its original background, *Triticum boeoticum*, was a 0;. The derived hexaploid lines used for most of this test resulted in a LIT of 2= to a fleck. No cultures were found with virulence on seedlings with this gene. However, in the field, *Sr*22 lines may be moderately susceptible.

***Sr*23**. This gene resulted in a LIT of 23C. Avirulence was associated with race 56-MBC and 151-QCB, -QFB, and QSH and a few cultures of recent aecial origin. Little variation of infection type was found among cultures avirulent on *Sr*23. Too few wheat lines were evaluated to determine the effect of host background. The sporulation area of the lesion increased slightly with increasing temperature.

None of the previous differentials had *Sr*23 in a combination which allowed its expression. In Selkirk, *Sr*6, 7b, 9d, and 17 all were epistatic to *Sr*23 and all provided resistance to most cultures that were avirulent on *Sr*23. Since 1969, only a trace of the isolates each year was identified as race 56 except in 1971, when 1% of the isolates were race 56. *Sr*23 was not detected in any commercial cultivar, although possibly it could have occurred undetected in some of the Selkirk derivatives. With cultures obtained from aecial spreads a LIT 2− occasionally was expressed by Exchange. This is thought to be caused by another gene, not *Sr*23.

***Sr*24**. The LIT produced was a 2− to a 2+. Changes in

temperature failed to affect the LIT; however, *Sr*24 usually resulted in a 2+ in Baart or Little Club background and a 2− in all other cultivars tested. A few cultures obtained from aecial spore spreads resulted in a LIT of 2+ regardless of the host being tested. Adult plants ranged from moderately resistant to moderately susceptible, the latter occurred when lines with *Sr*24 were grown near severely rusted material.

***Sr*25**. The LIT with this gene varied from a 2 to a 2−. All cultures tested were avirulent on seedlings with this gene. The LIT was stable regardless of culture, temperature, or host used in the tests. Adult plants tended to range from moderately resistant to moderately susceptible, due to variation in environmental conditions that currently are unspecified.

***Sr*26**. The LIT with this gene was expressed as a ;2− and was stable throughout the range of cultures and temperatures tested. We found no cultures virulent on this genotype. Adult plants were moderately resistant to resistant.

***Sr*27**. The LIT was a 0; regardless of culture or temperature used. We found no virulence on lines with *Sr*27 either as seedlings or adults.

***Sr*28**. The LIT was expressed as a fleck for all avirulent cultures tested against lines with this gene. No change in LIT was observed with changes in temperature. Avirulence was limited to a few cultures of recent aecial origin.

***Sr*29**. The LIT generally was a 2− but on a few occasions with minor variations in temperature it was recorded as a 2. We have a few cultures for which the LIT was a 2+ on all *Sr*29 genotypes over a range of temperatures. We found no culture that was virulent on this genotype. This genotype was evaluated for the first time in this study in 1976. Adult plants with this gene are moderately susceptible.

***Sr*30.** The LIT varied from a 2− to a 2++. The LIT was unaffected by temperature and only slightly by cultures. However, the LIT was always a 2−2, on the Australian cultivar Festiquay and was 22++ on the cultivar Webster and its derivative, BtSr30Wst. We have a few cultures of race 11-RHR that are virulent on this genotype. In the field, host cultivars with *Sr*30 produce pustules of moderate size, but no field data are available on race 11-RHR. This gene was unique in that it confers resistance to all but race 11-RHR. This race has occurred in only trace frequency among the isolates that have been identified since 1972. Thus, currently *Sr*30 is more useful for screening bulk cultures than as a differential cultivar.

***Sr*Tt-1**. The LIT varied from 0 to X−. The immune infection type resulted with races 151-QSH, 56-MBC, 32-RSH, and some isolates of aecial origin. A;1+ to X− infection type was typical of races 151-QSH, 56-MBC, 32-RSH, and some isolates of aecial origin. A ;1+ to X− infection type was typical of races 151-QFB and -QCB. However, a third infection type, (4,0) which was expressed under certain conditions gave hypersensitive flecks which were visible resulting in a LIT (4,;). In these cases, the number of type 4 infections was less than those that occurred on the background host line without *Sr*Tt-1. This phenomenon was typical of races 15-TNM, 113-RKQ and many others. The 4,; infection type of *Sr*Tt-1 has been studied in more detail by Ashagari (4,5). We classified type 4,; or 4,0 as a high infection type in our studies. In an adult plant, this host gene results in a few large susceptible infections.

None of the previous differentials had *Sr*Tt-1. This gene provided resistance to 10–29% of the isolates annually since 1972. Avirulence was found in races 151-QCB, -QFB, and -QSH, and 56-MBC. The gene occurred in most of the soft red winter wheats from Indiana, and in some of the western wheats. Probably it was introduced into the breeding programs from Ill#1/ Chinese*2//*Triticum timopheevii*.

***Sr*Tt-2**. The LIT was a 0 to a fleck. No variation of infection type was observed among cultures tested and with changes in temperature. Host background effect was not evaluated, because only one line was evaluated to most cultures. No cultures were virulent on this host genotype. Segregation within the host line has been a continual problem in spite of constant reselection.

***Sr*Tt-3**. The LIT varied from a 0 to 0;1+C depending on the cultures and lines being evaluated. Testing was insufficient to determine the source of this variation. Virulence was common in North American cultures of standard race 15.

More testing is needed to assure that this stock only possesses a single gene for rust resistance, before determining its usefulness as a differential. We know of no cultivar with this resistance and none of the differentials used previously had this gene.

***Sr*Tmp**. The LIT varied from a 2− at 18 C to a 2= at 30 C. No consistent variation was found among cultures. Apparently this gene was present in many hard red winter wheats, but the lowest LIT occurred in wheats with the Triumph-type background. In other winter wheats derived from the cultivar Turkey, a similar pattern of high and low infection types to a group of cultures was observed, but the infection type observed was always higher, 2+3.

No early data are available for *Sr*Tmp because it was not present in a differential cultivar. Since 1975, 62–86% of the isolates have been virulent on *Sr*Tmp. Virulence, however, existed only in the standard races 15 and 56. This gene has occurred in commercial cultivars of hard red winter wheat to some extent since 1874 when cultivar Turkey was introduced. It is of interest that the major epidemics since barberry eradication have been caused by standard races 56 and 15. Both of these races were virulent on *Sr*Tmp, a gene used throughout the southern Great Plains of the USA where stem rust may overwinter.

***Sr*LC**. The LIT was 2−. Avirulence was found only among isolates of aecial origin. This gene occurs in Little Club, Baart, and other lines used as backgrounds for other *Sr* genes.

***Sr*Gt**. The LIT was expressed as a 2 with the Australian cultivar Gamut which has other genes for seedling resistance. The BtSrGtGt-derived line had a LIT of 2+. No cultures produced a high infection type on lines with *Sr*Gt. No variation in infection type due to culture or temperature were observed.

***Sr*dp-2**. The LIT was generally a 2 when the host background was of a durum, tetraploid type. A LIT of 2++ resulted when the derived hexaploid was tested with the same cultures. No major difference in the LIT was caused by changes in temperature or culture.

The gene *Sr*dp-2 was present in the supplemental differential cultivar Golden Ball. Virulence currently exists principally in races 151-QFB, -QCB, and -QSH. Thus, virulence has varied from 10 to 31% of the isolates tested during the past 6 yr. This resistance gene is probably present in some of the durum wheat cultivars. Epistasis by *Sr*9e, and 13, and other undetermined genes may obscure its presence. This gene would be useful in a differential.

***Sr*X**. This gene has not been described previously but is presence was probably noted as early as 1928 when races 58, 62, and 63 were described (39). The LIT varied from a 23C to XC, depending on environmental conditions and host background. The Marquis backcross lines from Canada exhibited a lower infection type (XC) than the cultivar Marquis (23C) that historically has been used at the Cereal Rust Laboratory. When *Sr*X was in combination with other resistance genes, the chlorosis often was expressed even though the LIT associated with the other gene was epistatic to *Sr*X. For example, the LITs for lines with *Sr*29 was expressed as a 2−, with *Sr*X, a 23C and a derivative of the two host lines with both *Sr*29 and *Sr*X, 2−C. Insufficient data exists to detect differences in infection type caused by temperature and culture with this range of infection types.

The gene *Sr*X occurred in Marquis, one of the standard differential cultivars. Data on race prevalence indicated avirulence on lines with this gene as early as 1928. However, it was not until 1969 that race 113 was commonly found in the USA. Races 113-RTQ and -RKQ as well as 151-QCB and -QFB were avirulent on lines with *Sr*X. The latter two races were detected as early as 1965, but *Sr*7b (also present in Marquis) was epistatic to *Sr*X. The precentage of isolates virulent on lines with *Sr*X since 1972 has varied from 82 to 92. Lines are being increased so that this genotype can be used as a differential.

***Sr*McN**. The LIT was a 2−;, and avirulence was confined to a few cultures of aecial origin. This gene was found during the course of these studies when McNair 701 was used as a susceptible host on which to increase inoculum due to its resistance to leaf rust and powdery mildew. Gene *Sr*McN differs from *Sr*LC and currently is

thought to occur in a limited number of wheats. The variation in LIT was not studied.

***Sr*Kt'2'**. The LIT was a 2 regardless of the temperatures or cultures used. Infection type did not differ between Kota and the W2691-derived line.

The resistance provided by *Sr*13, 22, 24, 25, 26, 27, 29, Gt, and Tt-2 was 100% effective, making these genes useless in differential cultivars. However, they are possible sources of resistance for use in wheat breeding and they can be used to screen bulk cultures for detection of new sources of virulence.

In Table 3, the average infection types observed are compared with the infection types initially reported. Major differences were found in infection types with *Sr*7a, *Sr*24, and *Sr*25. In addition, a second LIT was recognized with *Sr*5 and *Sr*10, probably due to differences in host background. The second LIT recognized with *Sr*9a, 11, and Tt-1 was due to a difference among cultures. Lines with *Sr*6, 10, 15, and 17 previously were reported to become more susceptible with increasing temperature. We observed the opposite effect with *Sr*13 and to some extent with *Sr*9b.

To be useful as a differential cultivar a line should be susceptible to only a portion of the pathogen population . Data from this study show that the genes for race-specific seedling resistance could be classified in three groups based on virulence in the pathogen population that existed away from barberry. Group 1 host resistance genes (*Sr*9f, 9g, 16, 18, 19, 20, 28, LC, McN, and Kt'2') were susceptible to all or nearly all cultures studied, those of group 2, (*Sr*5, 6, 7a, 7b, 8, 9a, 9b, 9d, 9e, 10, 11, 12, 14, 15, 17, 21, 23, Tt-1, Tt-3, Tmp, dp-2, and X) were differential in response, and those of group 3 (*Sr*13, 22, 24, 25, 26, 27, 29, 30, Tt-2, and Gt) were 'universally' resistant, or nearly so.

In any study of physiological races, maintenance of historical and international continuity is useful. Therefore, the choice of differentials should be made accordingly. In the USA, race identification of the wheat stem rust pathogen historically had depended upon use of the standard and supplemental differentials. The genes for rust resistance in these lines, so far as is known, is shown in Table 4. *Sr*5, 7b, 9d, 9e, and 21 were the *Sr* genes present in the standard differentials that most frequently responded differentially. Although *Sr*14 may occur in Khapli, the LIT incited by the cultures used is due to another gene. Among supplemental differential cultivars, genes for resistance (*Sr*6, 7a, 7b, 8, 9b, 10, 11, 17, 23, and dp-2) responded differentially. *Sr*9a, 12, 14, 15, Tt-1, Tt-3, Tmp, and X, which were tested in this study, also responded differentially.

TABLE 4. Genes for stem rust resistance in the standard and supplemental differential cultivars of wheat used to determine physiologic races of *Puccinia graminis* f. sp. *tritici* in the USA

Cultivar	CI[a]	*Sr* gene(s)[b]	References
Standard differentials			
Little Club	4066	LC	25
Marquis	3641	7b,18,19,20,X	3,7,23,25,34
Reliance	7370	5,16,18,20	1,3,6,7,25,37
Kota	5878	7b,18,28,Kt'2'	7,23,25
Arnautka	1493	9d,+	25
Mindum	5296	9d,+	25
Spelmar	6236	9d,+	25
Kubanka	1440	9g,+	25
Acme	5284	9g,+	25
Einkorn	2433	21	11,25
Vernal	3686	9e	28
Khapli	4013	7a,13,14	25,42
Supplemental differentials			
Lee	12488	9g,11,16	11,25
Selkirk	13100	6,7b,9d,17,23	32
Bowie	13146	6,8	27
Golden Ball	6227	dp-2	10,37
Yuma	13245	9e,+	
Kenya Farmer	12880	7a,9b,10,11	12,13,15,17
Frontana-Kenya 58-Newthatch	13154	5,6,7a,8,9b	20

[a]CI = Cereal investigation number.
[b]The symbol + = additional gene(s) present for seedling resistance to wheat stem rust.

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